South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 1 September 1956
Weekly 2s. 6d.
Vol. 30 No. 35
Kaapstad, 1 September 1956
Weekliks 2s. 6d.

DOES DISSEMINATED SCLEROSIS OCCUR IN THE BANTU?

S. KRAMER, M.B., B.CH., B.SC. (RAND)
Medical Registrar, Baragwanath Hospital
R. LIPSCHITZ, M.B., B.CH. (RAND), F.R.C.S. (EDIN.)
Neuro-Surgeon, Department of Surgical Neurology, Baragwanath Hospital
M. B. SCHWARTZ, M.B., B.CH. (RAND), F.R.F.P.S. (GLASG.)
Physician, Baragwanath Hospital

M. Gelfand[1] states that disseminated sclerosis does not occur in the Bantu. F. M. K. Muwazi and H. C. Trowell[2] in their comprehensive review of Neurological Disease among African Natives of Uganda, state 'we have never encountered any of the demyelinating diseases of the nervous system. It is true a few cases of disseminated sclerosis have been reported in Africans in West Africa, yet the case records and investigations were somewhat restricted and the autopsy material scanty'.

B. Goldstein[3] reported 2 cases in East African Natives, one disseminated in type, the other of the progressive spastic paraplegic type seen in middle-aged persons.

Craig Cochran[4] reported a case in a non-European female of Xosa and Coloured extraction.

CASE RECORD

The following is the case record of a patient, of pure Bantu descent (daughter of a Zulu father and a Msutu mother) who presents the clinical picture of disseminated schlerosis.

M.M., aged 26, employed in a bakery, was first admitted on 26 July 1955 with the following complaints:

For a month before admission she had been walking 'like a baby'. She had noticed that on looking to the right she saw double and on turning her head to the right she experienced dizziness. This dizziness would occasionally cause her to fall to the ground. There had been a tendency over the previous month to drop objects, more so from the left hand.

Before this the patient had been quite well, except that over the past year she had occasional headaches, which if anything had tended to diminish in severity and frequency.

The findings on examination were: A fine nystagmus to the right and a coarser one to the left; diminished corneal reflex on the left. The left limbs were hypotonic, with a marked intention tremor and incoordination of alternating movements in the left hand. The patient showed similar cerebellar signs on the right, but much milder in degree.

The remainder of a detailed neurological examination was entirely negative.

The following investigations were carried out soon after admission:

X-rays of chest and skull were negative.

Full blood-count showed no abnormality.

Lumbar puncture; pressure 150 mm. of water and a satisfactory response to jugular-vein compression; cells—2 lymphocytes per c.mm.; chlorides 690 mg.%, sugar 44 mg.%, protein 118 mg.%; Wassermann reaction negative.

Wassermann reaction of the blood was negative.

During the next few weeks while in hospital the patient complained of precipitancy.

On 19 August a vertebral angiogram showed no displacement of the vessels and no abnormal filling defects. Shortly afterwards her condition began to improve and by the end of August she no longer complained of diplopia or dizziness and the cerebellar signs, though still present, were greatly diminished.

In October the cerebellar signs again became marked and in addition the patient developed totally new symptoms and signs. She complained of excessive salivation and dysphagia and her speech was markedly slurred. The excessive salivation and dysphagia subsided after a week, but the slurring of speech became worse. There was weakness of both lower limbs, so much so that she could not support herself. Both the knee and ankle jerks were greatly increased, ankle clonus being elicited on the left side; the spasticity was more marked on the left side. Gradually the spastic paraparesis regressed, but at the end of November the patient developed a marked weakness and spasticity of the whole of the left side, more marked in the face than the arm, and in the arm than the leg. By the end of December the spastic hemiparesis of the left side was the dominant lesion.

On 19 November an air encephalogram was carried out and showed that the fourth, third and lateral ventricles and the aqueduct of Sylvius were of normal size, shape and position. In none of the pictures were there any abnormal filling defects. On

26 November audiogram and caloric tests were carried out and found to be normal.

The patient again improved and towards the end of January 1956 she was discharged. At this stage her signs and symptoms were decreasing and she was just barely ambulant with help. Except for a few days in December during which the right pupil was found to be smaller than the left (but both pupils reacted to light and accommodation) she had developed no further signs.

The patient was readmitted for follow-up on 5 April 1956 when the findings were as follows: Slurring of speech, nystagmus to the right as well as to the left—coarse in both directions. A fine vertical nystagmus on upward gaze. The corneal reflexes were now brisk and equal on both sides. There was bilateral spasticity and cerebellar signs, both more marked on the left than on the right.

Now, however, in addition to these signs there was a loss of joint position sense in the right big toe. This sensory abnormality disappeared completely towards the end of April and about this time the inequality of the pupils, as previously described, reappeared for a few days.

Throughout the patient's stay in hospital her mental state was one of euphoria.

At the time of her discharge in the middle of May 1956 she was able to walk the length of the ward by means of holding on to beds or using crutches.

Repeated cerebrospinal-fluid studies showed negative Wassermann reactions, protein which ranged from 110 to 150 mg.%, and a negative colloidal-gold curve.

DIFFERENTIAL DIAGNOSIS

'Moderately severe mixed case is likely to be confounded only with diseases whose morbid processes are diffused; and of these cerebro-spinal syphilis alone need be considered. Clinical resemblance between the two is often intimate, but a history of the latter may be forthcoming, and in any case laboratory tests suffice to distinguish'.[5]

Against a diagnosis of acute disseminated encephalomyelitis are the absence of a history of vaccination or any infectious fever, the bright intelligence as against the post-encephalic 'vegetable', and the dissemination in time and place. In the non-European bizarre nervous presentations of avitaminosis are always to be considered.[6] This patient was extremely well nourished on admission and the dissemination in time and place occurred while she was in hospital on a well balanced diet.

Tumour infiltrating the base of the brain may simulate disseminated sclerosis.[7] The normal CSF pressure with satisfactory response to jugular-vein compression, the absence of increased intracranial pressure, and the normal findings of the special investigations, are against this diagnosis; as is the course of the disease as described in our patient.

Two points differ from the text-book description of disseminated sclerosis, viz. (1) the negative colloidal-gold curve, and (2) the protein in the CSF ranging from 110-150 mg.%. Nevertheless, these differences are compatible with the diagnosis of disseminated sclerosis.

Differences in incidence of positive colloidal-gold curves result from minor differences in technique. In the City Hospital of New York a 60-70% incidence of a positive colloidal-gold curve was obtained in disseminated sclerosis;[8] whereas at the Neurological institute of New York the incidence was 10-20%. It is generally agreed that the percentage of positive curves in routine clinical laboratories in disseminated sclerosis is low.[8]

McAlpine[9] in 512 cases found a protein greater than 100 in 5 cases. A protein as high as 255 has been recorded in disseminated sclerosis.[10] A high protein is found in 32% of cases in the initial stages of the disease.[10] Picht, Seuberling, Gaupt and Schroeder found the percentage of CSF abnormalities to be directly proportional to the severity of the clinical picture.[10] The high CSF protein in our case may well be associated with the rapidly progressive severity of the condition.

SUMMARY

A case is described in a young Bantu woman with a clinical picture typical of disseminated sclerosis. If the patient was a European no other diagnosis would have been entertained. In the Bantu, however, one accepts this diagnosis with reservation and awaits further developments.

SAMEVATTING

'n Klinies tipiese geval van verspreide sklerose in 'n jong Bantu vrou word beskryf. As dit 'n Blanke pasiënt was, sou hierdie diagnose algemeen aangeneem word. In die Bantu egter is die diagnose minder seker en moet 'n mens wag op verdere verwikkelings.

We wish to acknowledge thanks to Drs. S. M. Stone and G. W. Brammer of Germiston, who referred the case to us and kept us in contact with the patient. We wish to thank Dr. G. D. Elliot, Acting Superintendent of Baragwanath Hospital, for permission to publish this case.

REFERENCES

1. Gelfand, M. (1947): *The Sick African* (1944), p.246. Cape Town: Post-graduate Press and Stewart Printing Co.
2. Muwazi, F. M. K. and Trowell, H. C. (1944): E. Afr. Med. J., **21**, 2.
3. Goldstein, B. (1946): *Ibid.*, **23**, 170.
4. Cochrane, J. W. C. (1947): S. Afr. Med. J., **21**, 613.
5. Kinnier Wilson, S. A. (1940): *Neurology*, p. 175. London: Butler and Tanner Ltd.
6. Spillane, J. D. and Scott, G. I. (1945): Lancet, **2**, 261.
7. Miller, Z. R. (1955): Minnesota Med., **38**, 235.
8. Res. Publ. Assoc. Nerv. Ment. Dis., Vol. 28, p. 436.
9. McAlpine, D. (1955): Lancet, **1**, 1033.
10. Res. Publ. Assoc. Nerv. Ment. Dis., Vol. 28, p. 430.